

## Genetic and Environmental Influence on the Asymmetry of Dermatoglyphic Traits

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**ABSTRACT** Fluctuating asymmetry (FA) is defined as random deviations from bilateral symmetry of the body. Thus, its magnitude is often used to evaluate developmental homeostasis. In this study we evaluate the following hypotheses: 1) FA of dermatoglyphic traits has a significant genetic component; 2) prenatal maternal environment (PME) has a significant effect on the FA of dermatoglyphic traits in developmentally healthy individuals; and 3) genetic or environmental factors affect FA on organismal or systemic levels. Therefore, their effect is better seen in composite scores of FA rather than in FA indices for single traits. We analyzed 15 dermatoglyphic traits from 140 pairs of monozygous twins, 120 pairs of dizygous twins, and 106 pairs of mothers and daughters. All individuals were developmentally healthy. The influence of genetic and environmental factors on FA was evaluated by analysis of variance and regression analysis. For a majority of the traits in our study, FA showed significant but weak heritabilities, with values falling within the 0.20–0.35 range. None of the traits taken separately demonstrated the effect of PME on FA to be significantly greater than zero. The composite score of FA tended to have greater heritability values than individual traits. One of them, obtained in principal components analysis, showed a significant PME effect, supporting the hypothesis that FA is a systemic property. *Am J Phys Anthropol* 111:531–543, 2000.

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Fluctuating asymmetry (FA) is defined as small and directionally random deviations from bilateral symmetry. It is commonly used to evaluate developmental homeostasis, which includes the ability of the organism to buffer environmental and genetic perturbations and the ability to minimize random developmental errors (Van Valen, 1962; Zakharov, 1989; Palmer, 1994; Palmer and Strobeck, 1997; Livshits and Kobylansky, 1991; Fraser, 1994; Wilkins, 1997).

Since developmental errors are inevitable, no organism is perfectly symmetrical. A number of experiments have shown that

significant ontogenetic stress is associated with an increase in the number of developmental errors resulting in elevated FA (Mooney et al., 1985; Siegel and Smookler, 1973; Siegel et al., 1977). In humans, high levels of FA were documented for children whose mothers had poor health status (Kieser and Groenveld, 1994; Kieser et al., 1997) and among individuals diagnosed with some developmental disorders (Bar-

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den, 1980; Livshits et al., 1988; Rodewald and Chopra, 1991; Mellor, 1992; Goldberg et al., 1995, 1997a,b; Naugler and Ludman, 1996a, b; Thornhill and Møller, 1997; Kobylansky et al., 1997; Shapiro, 1983; Møller, 1997).

Twin studies have shown that increased dermatoglyphic asymmetry corresponds to a higher interpair variability in a number of behavioral tests as well as to a greater test-retest instability (Rose et al., 1987; Bogle et al., 1994a). Schizophrenia among discordant monozygous twins resulted in a greater interpair variability for FA of dermatoglyphic traits when compared to unaffected pairs of twins (Bracha et al., 1992), while the degree of asymmetry was related to clinical severity of disease (Mellor, 1992). Studies of dermatoglyphic traits are particularly valuable with respect to developmental stability. The unique quality of these traits is that once formed at the end of the first and beginning of the second trimester of embryonic development, they remain unchanged through the individual's life (Penrose and Ohara, 1973; Gooseva, 1986). Therefore, increased dermatoglyphic asymmetry can be related to a nonspecific distortion at an early stage of embryonic development.

While the influence of developmental environment on FA is well-accepted, the role of genetic factors in FA determination is debated. Results of experiments and observations on the heritability of FA of different morphological structures vary. Some studies have implicated an absence of any genetic component (Thoday, 1958; Potter and Nance, 1976; Leamy, 1997; Winding, 1998; Corruccini et al., 1988; Corruccini and Potter, 1981); others have found significant heritability values varying between 20–45% (Livshits and Kobylansky, 1989; Møller and Thornhill, 1997). Even within a developmentally related group of traits, such as dermatoglyphs, the data are controversial. The heritability of the FA for a–b ridge count was shown to be very close to zero (Arrieta et al., 1993; Bogle and Reed, 1997), while the analysis by Hirth et al. (1984) found limited genetic bases for the FA of interdigital ridge count. The FA for fingertip ridge counts and palm patterns ex-

pressed weak but significant heritability levels (Singh, 1970; Polukhov, 1984; Martin et al., 1982; Loesch and Martin, 1982), contrary to the study by Holt (1954) who implicated purely environmental determination for fingertip FA, based on interfamilial correlations. Another source of evidence implying some genetic bases for dermatoglyphic FA comes from well-established differences among populations (Jantz, 1975; Ditmar, 1998).

In the present study, we use samples of palm prints of monozygous and dizygous twin girls, as well as their mothers, to estimate and compare the role of genetic factors and developmental environment in FA determination. All individuals were developmentally healthy. Since dermatoglyphic traits remain unchanged after the second trimester of gestation, we can address the influence of prenatal maternal environment (PME) on the magnitude of FA. Twins, developing in the same uterus, are expected to share certain stress factors such as mother's sickness or poor maternal diet. On the other hand, sharing the same uterus can be a stress factor. Also, twins and singletons do not differ significantly by FA level (Markow and Gottesman, 1989); the placental proximity effect is known to increase the within-pair variation among monochorionic twins as well as twins with fused placentas (Bogle et al., 1994b). If PME plays a more significant role in FA determination than genetic factors, we would expect the mono- and dizygotic twins to have similar amounts of within-pair variation by FA. If, to the contrary, the effect of genetic factors is stronger, monozygotic twins should show a smaller within-pair variation than dizygotic twins.

## MATERIALS AND METHODS

### Samples and traits

We analyzed three samples comprised exclusively of females, including monozygous twins (140 pairs), dizygous twins (120 pairs), and mother/daughter pairs (106 pairs). Palm prints of these individuals were kindly provided by the Anuchin Anthropological Museum, Moscow State University, Russia. All palm prints were collected from

genetically healthy people, living in Moscow.

Palm and fingerprints were processed according to the method described by Cummins and Midlo (1961) and Penrose (1968). Fifteen dermatoglyphic traits were obtained from each print of the left and right hands, respectively. These traits were then used to calculate asymmetry indices:

- 1-5. *Ridge counts on each digit* (RC1-5) were scored as the number of ridges crossing the segment that connects the triradius and the center of the finger pattern. If more than one ridge count was available for a finger, as in the case of whorls or double loops, only the largest one was used for further analysis. The ridge count of arches was conventionally scored as zero.
6. *Total finger ridge count* ( $\Sigma RC$ ) was calculated as the sum of the ridge counts for five digits from the thumb to the fifth finger.
7. *The sum of radial triradii* (Rtr) was calculated as the sum of whorls, ulnar loops, and double loops from five digits on each hand.
8. *Total sum of triradii for five digits* ( $\Sigma tr$ ) was calculated according to equation (1).

$$\Sigma tr = U + R + 2*(D + W), \quad (1)$$

where  $U$ ,  $R$ , and  $D$  are the numbers of ulnar, radial, and double loops, respectively, and  $W$  is the number of whorls.

- 9-12. *Line A, B, C, and D exits* were determined as described by Cummins and Midlo (1961).
13. *Main palmar line index*, or *Cummins index* (Ic), was calculated as a sum of A and D exits (Cummins and Midlo, 1961).
14. *a-b interdigital ridge count* (a-bRC) was defined as the number of ridges that cross a line drawn between triradii a and b.
15. *Interdigital pattern score* (S) was calculated as the number of dermal ridges included in the interdigital patterns. If two ridge counts were available for the same pattern, only the maximal one was included.

## Statistical treatment

Since systematic interobserver error between families might artificially inflate the heritability estimates (Palmer and Strobeck, 1997), all palm prints were scored by the same observer (E.A.P.). Random error, if present, would result in somewhat lower estimates of heritability. Repeated scoring of the same set of 20 palm prints allowed us to estimate the reliability coefficients within the 96.4-99.8% agreement range for ridge counts, and the 95.8-96.6% range for main line exits.

The index of FA for each trait was calculated as the absolute difference between a trait on the right and left sides of the body:

$$FA_i = |(X_iR - X_iL)|, \quad (2)$$

where  $X_iR$  and  $X_iL$  are individual values for the trait on the left and right side of the body. Indices of FA obtained in Equation 2 were transformed in order to bring their distribution closer to normality by taking the natural logarithm of the sum of  $FA_i$  and 1.00. The minimum transformed index for any dermatoglyphic trait equals zero.

It is possible that the direction of asymmetry can be inherited. This could confound the value of the FA index, since the deviations from bilateral symmetry would be directionally nonrandom. To evaluate this possibility, we also analyzed the indices of directional asymmetry ( $DA_i$ ):

$$DA_i = X_iR - X_iL. \quad (3)$$

In order to weight the FA and DA indices equally and remove any correlation with the magnitude of the trait, the effect of size was linearly removed by regression. The indices of asymmetry were regressed on the sum of the trait from the left and right hands, and the standardized residuals were used in subsequent analyses. Hereinafter, the standardized residuals of FA and DA are referred to as  $FA'$  and  $DA'$  indices.

Indices of  $FA'$  do not correlate with the magnitude of a trait and have the same means, which allows us to sum them to create a total asymmetry index (TFA). Total fluctuating asymmetry was calculated as a sum of 15  $FA'$  indices for single dermatoglyphic traits, according to Equation (4):

$$\text{TFA} = \frac{1}{k} \sum_{i=1}^k \text{FA}_i, \quad (4)$$

where  $k$  equals 14, the total number of individual traits in our study. Total ridge count ( $\Sigma\text{RC}$ ), being a composite trait of traits 1–5, was not included in TFA.

### Kruskal-Wallis analysis of twin dyads

Since none of the dermatoglyphic traits has a continuous variation in the strict sense of the term and because of the marked departure from normality of the  $\text{FA}'$  indices, we used the Kruskal-Wallis nonparametric method of analysis of variance for a conservative test of significance. The null hypothesis was that variation in FA and DA among pairs is the result of random variation. This hypothesis was tested by creating two resampled FA groups by random drawing of individuals from other pairs. Two sets of resampled pairs, where each twin was paired with a randomly selected twin of the same zygosity, were created for the comparison with mono- and dizygous twins.

The within-pair difference for each sample was calculated as:

$$2 * |\text{FA1}'_i - \text{FA2}'_i| \quad (5)$$

and

$$2 * |\text{DA1}'_i - \text{DA2}'_i|, \quad (6)$$

where  $\text{FA1}'_i$  and  $\text{FA2}'_i$  are values of  $\text{FA}'$ , and  $\text{DA1}'_i$  and  $\text{DA2}'_i$  are values of  $\text{DA}'$  for trait  $i$  calculated for the first and the second member of the pair, respectively.

The within-pair difference in twin samples for each trait was compared to that of the control group by calculating an H-criterion with the Kruskal-Wallis procedure. The H-criterion is approximately distributed as  $\chi^2$  (Walpole and Myers, 1978).

Parametric analysis of variance and regression analysis were used to obtain estimates of heritability values.

### Analysis of variance

Analysis of variance was used to estimate broad sense heritability as:

$$h^2 = \frac{V_g}{V_p}. \quad (7)$$

Total phenotypic variance ( $V_p$ ) can be presented as a sum of three components:

$$V_p = V_g + V_{ef} + V_{ew}, \quad (8)$$

where  $V_g$  is genetic variance,  $V_{ef}$  is within-family environmental variance, and  $V_{ew}$  is within-group environmental variance. In our study,  $V_{ew}$  is associated with the PME variation among families. In our study, this component is due to differences in intra-uterine environment among twin pairs. These three components of phenotypic variance were estimated following the standard twin model of Haseman and Easton (1970), as modified by Christian et al. (1974, 1975). The application of this model is only warranted when total phenotypic variance within mono- and dizygous twins is equal. A smaller phenotypic variance among monozygous twins might result in overestimation of heritability (Corruccini et al., 1988). The significance of the variance difference among mono- and dizygous twins can be tested with the  $F'$  criterion (Christian et al., 1974):

$$F' = \frac{\text{AMS}_{(\text{MZ})} + \text{WMS}_{(\text{DZ})}}{\text{AMS}_{(\text{DZ})} + \text{WMS}_{(\text{MZ})}}, \quad (9)$$

where AMS is the mean square among pairs and WMS is the mean square within-pairs, and MZ and DZ are subscripts for mono- and dizygous twins, respectively.

Analysis of variance partitions AMS into two components: factorial ( $V_f$ ) and random ( $V_e$ ). Mean square within-pairs include only  $V_e$ :

$$\text{AMS} = 2 * V_f + V_e. \quad (10)$$

$$\text{WMS} = V_e. \quad (11)$$

When warranted by a nonsignificant  $F'$  criterion,  $V_f$  and  $V_e$  for mono- and dizygous twins are found as:

$$V_{f(\text{MZ})} = V_g + V_{ew} + 0.5V_{ef}. \quad (12)$$

$$V_{f(\text{DZ})} = 0.5V_g + V_{ew} + 0.5V_{ef}. \quad (13)$$

$$V_{e(\text{MZ})} = 0.5V_{ef}. \quad (14)$$

$$V_{e(\text{DZ})} = 0.5V_g + 0.5V_{ef}. \quad (15)$$

These equations show that while pairs of both dizygous and monozygous twins completely share a common uterine environ-

ment, the former share on average only half of the genetic material.

The broad sense heritability is by definition the ratio of genetic variance over total phenotypic variance. Combining Equation 7 with 10–15, two alternative estimates of broad-sense heritability can be derived:

$$h^2 = \frac{4 * (WMS_{(DZ)} - WMS_{(MZ)})}{TMS} \quad (16)$$

$$h^2 = \frac{4 * (AMS_{(MZ)} - AMS_{(DZ)})}{TMS}, \quad (17)$$

where  $TMS = 0.5(AMS_{(MZ)} + WMS_{(MZ)} + AMS_{(DZ)} + WMS_{(DZ)})$ —an average total mean square for two samples.

These estimates can be tested by F criteria (Christian et al., 1975; Corruccini et al., 1988):

$$F = \frac{WMS_{(DZ)}}{WMS_{(MZ)}} \quad (18)$$

and

$$F = \frac{AMS_{(MZ)}}{AMS_{(DZ)}}. \quad (19)$$

Equations 11–16 allow us to estimate two other components of phenotypic variance:

$$\frac{V_{ew}}{V_p} = \frac{AMS_{(DZ)} - 3WMS_{(DZ)}}{TMS} \quad (20)$$

and

$$\frac{V_{ef}}{V_p} = \frac{4 * WMS_{(MZ)}}{TMS}. \quad (21)$$

### Principal components analysis

Genetic or environmental factors are expected to affect developmental homeostasis on an organismic or systemic level. Therefore, the composite score of FA may be a more adequate measure of developmental disturbances than FA index for any single trait. The average fluctuating asymmetry, TFA, is a candidate proxy for the systemic FA indicator. Another possible general indicator of FA is one that is based on a weighted sum of the traits. The weights can be obtained as regression coefficients of the trait's score on a factor score. Factor scores from principal components analysis are based on correlations among the indices.

Principal component analysis was performed on FA' indices of pooled MZ and DZ twins. In the context of our study, principal components (PCs) are used as a linear transformation of an original set of variables in order to reduce dimensionality. Since the eigenvectors of a product moment matrix are real-valued, the principal components analysis does not require any assumption of bivariate normality (Green, 1976; Gower, 1966). In our case, univariate normality was approached by logarithmic transformations of FA indices. The composite measures of FA were obtained as factor scores of the first and second PCs. These measures were evaluated according to the twin model described in Analysis of Variance, above.

### Regression analysis of mother-daughter pairs

Indices of FA' and DA' for mothers were regressed on the values of FA' and DA' for their daughters. All 15 dermatoglyphic traits plus their sum, TFA, were analyzed. The narrow-sense heritability, which is the ratio of the additive component of genetic variance ( $V_A$ ) to total phenotypic variance ( $V_p$ ), was calculated, following Falconer (1960), as:

$$\frac{V_A}{V_p} = 2 * b, \quad (22)$$

where  $b$  is a regression coefficient in the equation predicting the child's asymmetry, based on the magnitude of the mother's asymmetry.

## RESULTS

### Nonparametric analysis of twin samples

The results of nonparametric analysis of within-pair variation are summarized in Table 1. We used the Kruskal-Wallis test of significance to compare the within-pair differences of each twin sample with the resampled groups formed from twins of the same zygosity. For the monozygous sample, 12 out of 16 traits showed significantly smaller within-pair differences for FA' at the 0.05 level than would be expected in randomly formed pairs. Six traits had H-criterion  $P$  values less than 0.01. These dif-



TABLE 1. *H*-criteria for Kruskal-Wallis comparison of asymmetry indices of monozygotic and dizygotic twins with the randomly formed pairs<sup>1</sup>

Trait	FA'		DA'	
	MZ	DZ	MZ	DZ
RC1	01.99	00.05	00.15	00.04
RC2	03.50*	01.99	00.24	00.00
RC3	04.76*	01.35	01.13	00.00
RC4	04.89*	00.48	00.45	00.34
RC5	04.20*	02.46	02.30	00.18
ΣRC	09.51**	07.07**	00.24	00.16
Rtr	07.08**	00.18	02.98	02.35
Σtr	08.16**	01.34	01.14	00.26
Line A-exit	00.05	00.00	02.57	01.19
Line B-exit	05.99*	00.19	04.15*	03.00
Line C-exit	15.98**	05.92*	04.32*	02.18
Line D-exit	16.30**	06.14**	06.20**	00.45
Ic	05.41*	05.41*	05.14*	02.31
a-bRC	00.30	00.12	00.00	00.03
S	00.98	00.30	01.55	00.12
TFA	26.14**	08.00**		

<sup>1</sup> FA', fluctuating asymmetry index; DA', directional asymmetry index.

\*  $P < 0.05$ , 1 df.

\*\*  $P < 0.01$ , 1 df.

ferences might be a result of sharing of either PME or genetic material, or both, by monozygous dyads. However, 10 of 15 dermatoglyphic traits did not show significant differences in FA' when the dizygous pairs were compared to a random set. Two composite scores of FA' (ΣRC and TFA) and FA' associated with main-line exits did demonstrate significant differences. H-criteria for both composite scores of FA' are particularly high, with  $P$  values below 0.01.

Random within-pair variation of FA' among dizygous twins indicates that genetic factors have only a mild influence on its magnitude, so that the amount of shared genetic material within dizygous dyads is insufficient to produce any significant difference from random fluctuations. These results suggest a prevalence of genetic factors in FA determination over those of PME factors. The fact that TFA produced lower  $P$  values than any of the traits taken individually for both samples supports the suggestion that FA is a systemic property.

The DA' of main-line exits also showed significant differences from the control group for MZ twins. Since the direction of asymmetry of these traits may be under the influence of genetic factors, it cannot be considered directionally random. This influence may lead to overestimation of the heritabil-

TABLE 2. Results of ANOVA analysis for twin samples<sup>1</sup>

FA'	$V_p$	$h^2 =$		$F_g$	$V_{ef}/V_p$	$V_{ew}/V_p$
		$V_g/V_p$	$V_e/V_p$			
RC1	1.00	0.12	1.14	0.84*	0.04	
RC2	0.98	0.14	1.17	0.81*	0.05	
RC3	0.99	0.22*	1.28	0.82*	0.00	
RC4	1.00	0.20*	1.28	0.71*	0.09	
RC5	0.99	0.28*	1.38	0.72*	0.00	
ΣRC	0.99	0.35*	1.54	0.65*	0.00	
Rtr	0.99	0.20*	1.25	0.80*	0.00	
Σtr	1.00	0.24*	1.31	0.76*	0.00	
Line A-exit	0.99	0.30*	1.43	0.70*	0.00	
Line B-exit	0.99	0.23*	1.30	0.77*	0.00	
Line C-exit	0.99	0.05	1.06	0.79*	0.16	
Line D-exit	0.99	0.04	1.05	0.79*	0.17	
Ic	0.99	0.05	1.06	0.80*	0.15	
a-bRC	1.00	0.10	1.12	0.84*	0.06	
S	0.99	0.14	1.16	0.86*	0.00	
TFA	1.00	0.29*	1.41	0.71*	0.00	

<sup>1</sup> Heritability values ( $h^2$ ) are presented as average between two estimates from Equations 17–18. For identification of variance ( $V$ ) measures, see text. FA', fluctuating asymmetry index.

\*  $P < 0.05$ , with 138 and 118 df.

ity of FA. We suggest that interpretation of the heritability of FA of these traits should be approached with caution.

Once significance of variance differences was evaluated by a nonparametric analysis, we proceeded with parametric analysis of variance.

### Broad-sense heritability estimates of FA from twin samples

Criteria of  $F'$  varied from 1.12–0.92 with 138 and 118 degrees of freedom, and detected no significant differences in the total phenotypic variance between MZ and DZ twin samples for any trait, allowing us to proceed with heritability estimates.

Table 2 presents broad-sense heritability estimates obtained by averaging the two heritability estimates from Equations 16 and 17. Nine out of 16 FA' indices in the analysis produced weak, but statistically significant, estimates that vary from 0.20 for RC4 to 0.35 for ΣRC (Table 2).

Heritability indices of FA for finger-ridge counts tended to increase from thumb to fifth finger, so that heritabilities of FA' for RC1 and RC2 were not significant, and FA' of RC5 had the highest significance. Heritability estimates were also significant for FA' of line A and B exits. As noted before, the FA heritability for these lines may be overestimated because of the presence of heritable

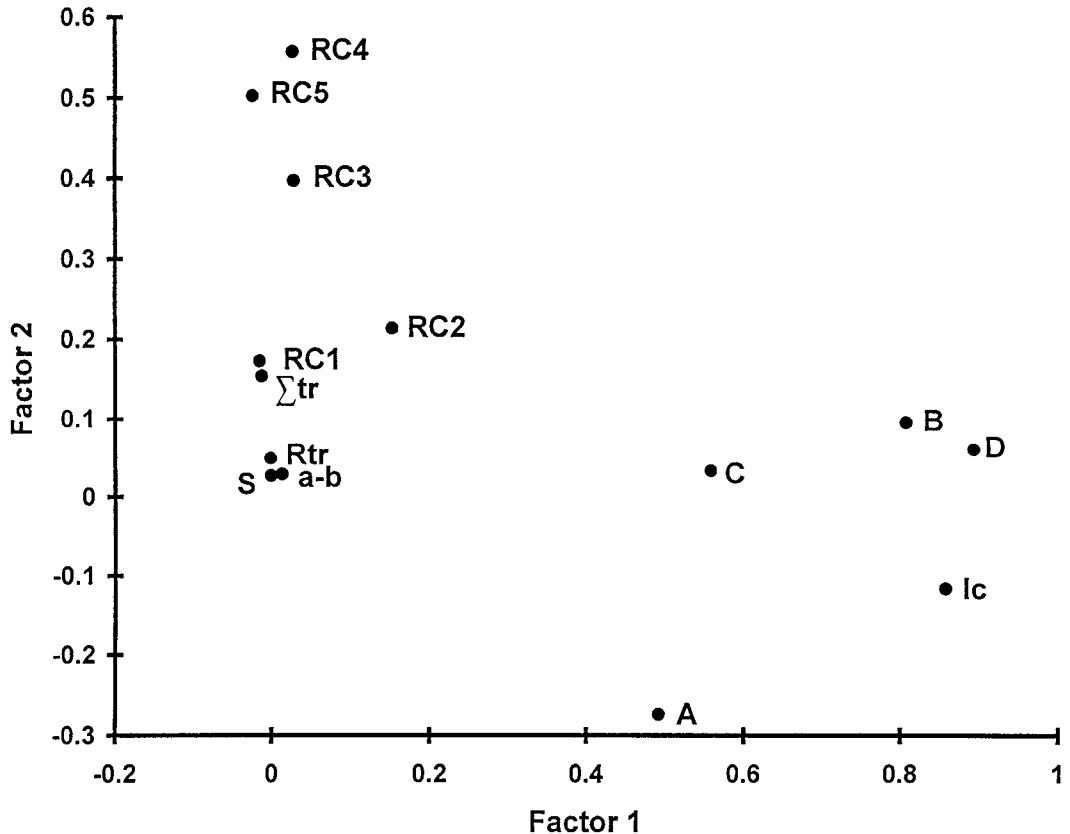


Fig. 1. Factor loadings in principal components analysis. Factor 1 vs. factor 2.

DA. The traits on the interdigital pads (a–b ridge count and interdigital pattern score) showed a complete absence of genetically determined variation. The total fluctuating asymmetry index had the second highest heritability level, after  $\Sigma RC$ .

Without exception, the traits demonstrated  $V_{ew}$  indistinguishable from zero, suggesting that the among-pair variation in our samples cannot be explained by the PME differences and comes exclusively from genetic variation. However, for the FA' indices associated with the main-line exits, these estimates were higher than those for other traits.

#### Principal components analysis of FA

A composite score of FA was produced from factor scores obtained in principal components analysis. The loadings produced by the first four PCs are summarized in Fig-

ures 1 and 2. The first four principal components account for 51.2% of the phenotypic variation. Low eigenvalues might be expected as a result of the inherent indeterminacy of FA measurements (Whitlock, 1996).

Fluctuating asymmetry indices associated with main-line exits had high positive loadings on the first PC, while the second PC loaded on the FA' of finger-ridge count. Fluctuating asymmetry indices for main-line exits tend to have opposite correlations to those of the rest of the traits. Particularly high loadings on PC2 were obtained by FA of those fingers that had significant broad-sense heritability estimates (see Fig. 1). The discrimination of palmar and finger FA indices by first and second PCs implies two independent factors controlling their magnitude.

Traits whose FA' received low or insignificant heritability values (Table 2) tended to

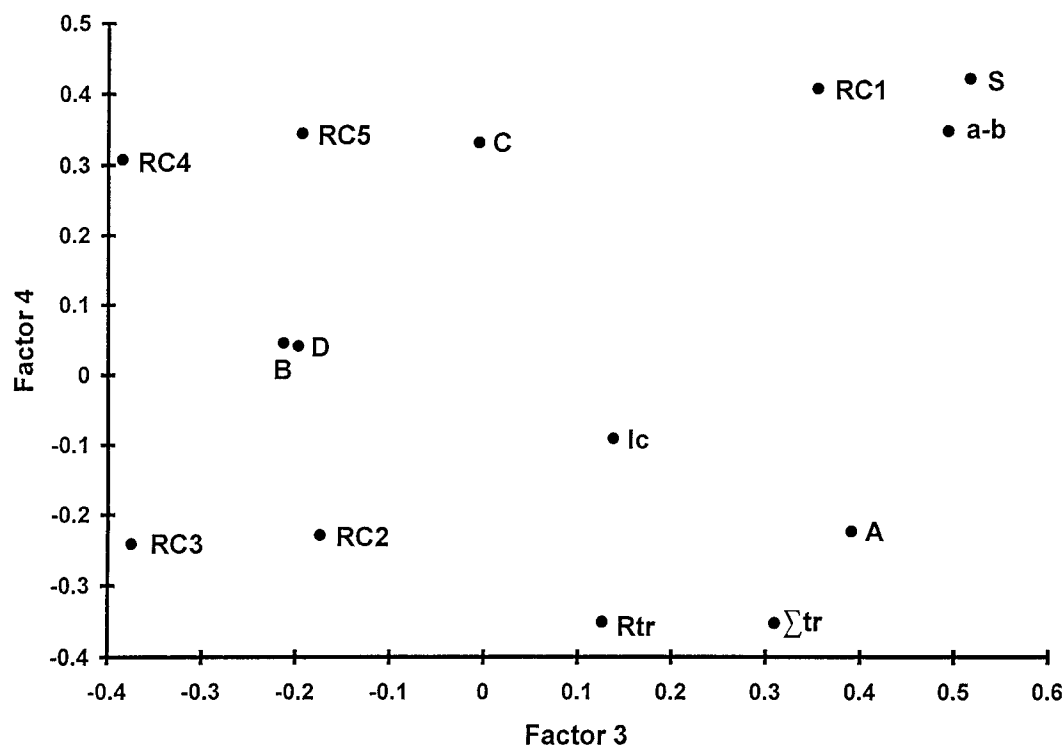


Fig. 2. Factor loadings in principal components analysis. Factor 3 vs. factor 4.

TABLE 3. Results of ANOVA analysis of the factor scores derived from the principal components analysis

Principal component	% total variance	$V_g/V_p$	$F_g$	$V_{ef}/V_p$	$V_{ew}/V_p$	$F_{ew}$
PC1	22.83	0.02	1.03	0.70*	0.28*	1.38
PC2	12.10	0.36*	1.49	0.64*	0.00	1.00
PC3	8.14	0.30*	1.38	0.70*	0.00	1.00
PC4	8.08	0.18	1.22	0.82*	0.00	1.00

<sup>1</sup> For identification of variance (V) measures, see text.

\*  $P < 0.05$ , with 138 and 118 df.

have zero loadings on the first two PCs, and high loadings on the third and fourth (see Fig. 2). This is true for the FA of a-b interdigital ridge count, interdigital pattern score, and ridge counts for the first fingers.

Variance analysis of composite scores obtained by the first and second PCs yielded significant heritability estimates for the second and third PCs but not the first and fourth (Table 3). The first PC had a  $V_{ew}$  component that was responsible for 28% of phenotypic variation, suggesting a significant effect of PME on the variation of the first PC.

#### Narrow-sense heritability estimated from mother/daughter pairs

We applied regression analysis to the mother/daughter pairs in order to measure narrow-sense heritability (Table 4). Recall that narrow-sense heritability measures additive genetic variance only, and therefore is expected to be lower than broad-sense heritability.

Only six heritability estimates obtained in the regression analysis were statistically significant, all falling within the 0.21–0.40 range. The narrow-sense heritabilities of FA' for finger-ridge counts expressed tendencies similar to those of broad-sense ones. Heritability estimates of FA' for RC4 and RC5 were greater than those for RC1 and RC2. Interdigital patterns show FA' heritability to be very close to zero in both methods. Pearson product moment correlation of the heritability estimates obtained in regression and analysis of variance was 0.55 (see Fig. 3), demonstrating a remarkable



TABLE 4. Results of regression analysis of mother/daughter pairs<sup>1</sup>

Trait	FA'		DA'	
	$V_A/V_p$	SE	$V_A/V_p$	SE
RC1	0.24*	0.20	0.00	0.21
RC2	0.00	0.21	0.04	0.20
RC3	0.12	0.20	0.00	0.24
RC4	0.26*	0.20	0.00	0.20
RC5	0.26*	0.18	0.01	0.20
ΣRC	0.34*	0.06	0.00	0.21
Rtr	0.02	0.80	0.00	0.23
Σtr	0.06	0.63	0.00	0.20
Line A-exit	0.21*	0.16	0.12	0.28
Line B-exit	0.18	0.20	0.24*	0.20
Line C-exit	0.16	0.38	0.18	0.19
Line D-exit	0.18	0.26	0.19*	0.18
Ic	0.08	0.21	0.22*	0.17
a-bRC	0.00	0.20	0.02	0.24
S	0.00	0.20	0.00	0.20
TFA	0.40*	0.15		

<sup>1</sup> FA', fluctuating asymmetry index; DA', directional asymmetry index;  $V_A$ , additive variance;  $V_p$ , phenotypic variance.

\*  $P < 0.05$ , with 105 df.

reproducibility of these estimates, by the two methods in two different samples.

Contrary to expectation, five individual traits in our analysis as well as TFA analysis yielded narrow-sense heritability estimates higher than the broad-sense ones. We suggest that the influence of PME on these traits results in elevated similarities between mothers and daughters that produces overestimated narrow-sense heritability estimates. A similar effect of PME on FA was observed in prematurely born children (Livshits et al., 1988).

Narrow-sense heritability for DA indices was found to be insignificant for the majority of traits. However, it tended to be higher among those traits associated with the main-line exits, with three estimates reaching the level of significance. The tendency for most main-line exits to have a heritable DA was also supported by a Kruskal-Wallis test of twins (Table 1). The heritability estimates for all DA and FA values obtained from regression analysis had zero correlation ( $r = 0.06$ ), which does not support a direct influence of DA genetic factors on FA. Whether heritability of DA can indirectly affect the heritability estimates of FA for any specific trait requires further study.

## DISCUSSION

The dermatoglyphic traits we studied divide into three groups corresponding to the

pattern of covariation summarized by PCs. 1) Palmar-line exits and their derivative Ic exhibited high positive loadings on the first PC. Both fluctuating and directional asymmetries of these traits have some genetic basis. What is more important, FA' of these traits tends to have nonzero PME effect, as suggested by narrow-sense heritability estimates exceeding the broad-sense ones and  $V_{ew}/V_p$  ratios obtained in analysis of variance. When these traits are combined within the first PC, the PME effect reaches the level of significance and includes 28% of the phenotypic variance. On the other hand, the heritability of DA' for these traits can result in the overestimation of both genetic and PME effect on FA. 2) The second group includes the traits associated with fingertips, whose FA' loaded on the second PC. These traits exhibited significant heritability estimates for FA' in both the narrow and broad senses, with values that tended to increase from thumb to fifth finger. Similar heritabilities of the FA for finger-ridge counts were obtained in other studies and varied between 20–44% among different pairs of relatives in three other family studies (Singh, 1970; Polukhov, 1984; Martin et al., 1982). Directional asymmetry of these traits is not inherited. 3) This group includes two traits derived from interdigital ridge counts a-bRC and S that did not load on the first two PCs but loaded highly on the third and fourth. These traits had particularly low heritability estimates for either FA' or DA' indices in our analysis, in agreement with data reported by Arrieta et al. (1993) and Bogle and Reed (1997), rather than with those by Hirth et al. (1984). The ridge count on the thumb, that also predominantly loaded on the third and fourth PCs, can be attributed to this group (see Fig. 2).

Some part of the explained variation in FA was expected to be associated with differences in maternal environment. To the contrary, we find that the role of PME is not significant for FA' of any of the traits studied separately. The composite score obtained from the first PC (Table 3) is the only instance where the effect of PME was significant. A stronger role of PME has been demonstrated in studies from individuals experiencing high developmental stress

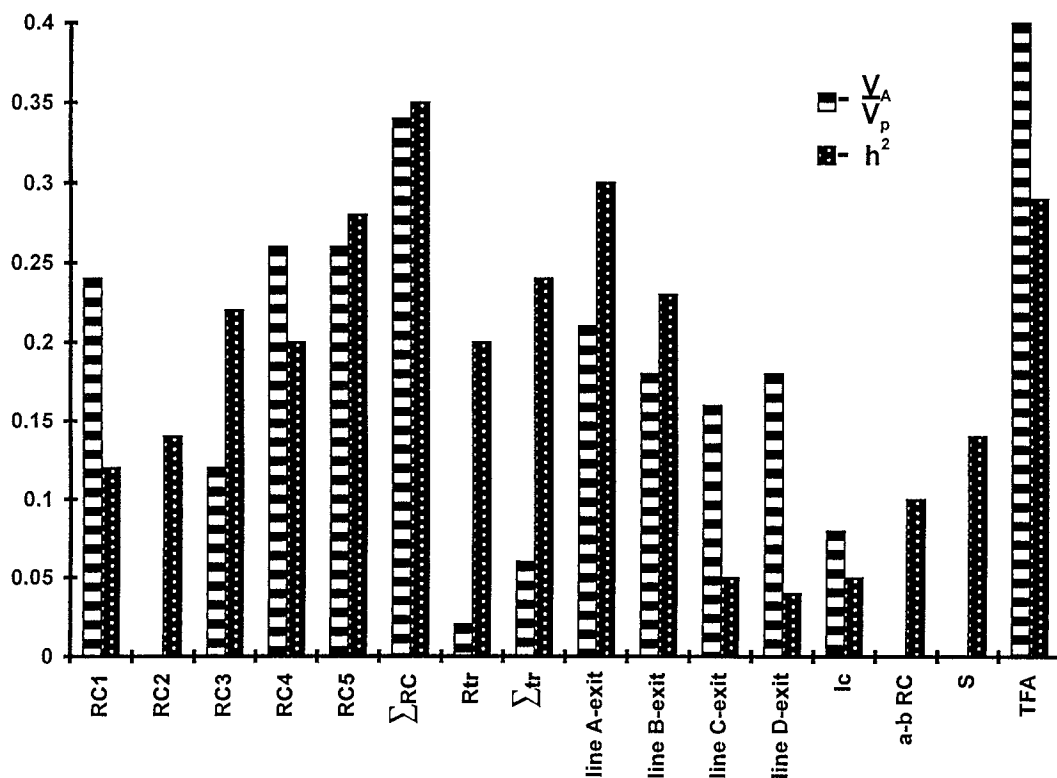


Fig. 3. Comparison of broad- and narrow-sense heritability. Correlation = 0.55.

(Kieser et al., 1997; Livshits et al., 1988). Children from mothers who smoke or are obese had elevated FA (Kieser et al., 1997). However, large differences between risk and control groups were observed only with the combination of the two risk factors. The level of PME influence on FA' observed in our study suggests that the variation in developmental environment among healthy twins was insufficient to produce any noticeable effect on separate FA scores. On the other hand, the competitive influences of twins on each other could increase the variation within some pairs of twins (Bogle et al., 1994b; Corey et al., 1979) and overshadow the PME effect.

The greatest portion of FA' variation cannot be explained by either genetic or PME factors. Unexplained variation might also arise from random developmental or measurement errors, as well as of environmental factors that are beyond the control of this

study, such as interpair differences in placental attachment and embryonic position.

Another source of unexplained variation may be the low developmental repeatability of FA (Whitlock, 1996). The low repeatability of FA results from the equal probability that traits on the left and right sides will deviate in the same or opposite direction from the inherited value. Consequently, if an individual organism had been given additional chances to develop under the same environmental conditions, the FA would not arrive at the same value each time. An example is helpful in order to illustrate the low repeatability of FA. One can imagine a factor  $f$ , that can be either genetic or environmental, which causes the trait on both the left and right hand to deviate from the genetically predetermined value. The magnitude of this deviation is a function of the value of this factor. An organism under an influence of the  $f$ -factor has four equally

TABLE 5. Influence of a hypothetical factor  $f$  on the level of FA<sup>1</sup>

$f = 1$	FA	$f = 2$	FA	$f = 3$	FA
$R + 1$	0	$R + 2$	0	$R + 3$	0
$L + 1$		$L + 2$		$L + 3$	
$R - 1$	2	$R - 2$	4	$R - 3$	6
$L - 1$		$L - 2$		$L - 3$	
$R + 1$	2	$R - 2$	4	$R - 3$	6
$L - 1$		$L + 2$		$L + 3$	
$R - 1$	0	$R - 2$	0	$R - 3$	0
$L - 1$		$L - 2$		$L - 3$	
Average FA	1		2		3

<sup>1</sup> The rows can be considered four equally probable ways of development for a single trait or a single hypothetical response for each of four individual traits to the factor's influence.  $L$  and  $R$  are genetically designated values of the trait on the left and right side, correspondingly. FA is the fluctuating asymmetry index.

probable routes of development for every trait:  $R + fL + f$ ,  $R + fL - f$ ,  $R - fL + f$ , and  $R - fL - f$ , where  $R$  and  $L$  are the values of the trait on the right and left hands that would develop in the absence of the factor (Table 5). Only two of these alternatives will result in an FA value different from zero. Therefore, while a nonzero FA always indicates the influence of an  $f$ -factor, a zero FA index does not signify its absence. When a single trait is studied for FA, only half of the individuals under the influence of the same  $f$ -factor will produce FA different from zero. However, if several traits are affected by the same factor, the average FA calculated for a number of traits should approach the magnitude of that factor (Table 5).

In our study, the composite scores  $\Sigma RC$  and TFA, as well as PC2 and PC3, produced significant heritability values that are higher than average for the individual traits (Tables 2 and 3), as was expected. The PME effect was statistically significant only in the factor score of the first PC. An argument has been made (Livshits et al., 1998) that FA cannot be considered a systemic property due to the low correlations among FA indices for single traits. As was shown above, the weak correlations among FA of individual traits are expected according to the low repeatability of FA. In our study, the covariation among the FA' of single traits was sufficiently patterned to produce factor scores with both significant heritability and PME effects. Therefore, we propose that the larger amount of explained varia-

tion in the first PC than in the FA of single traits supports the hypothesis that FA is a systemic property.

In the light of the inherently low repeatability of FA, our heritability estimates appeared to be unexpectedly high. A model based on the assumption of normality of the distribution of traits estimates the maximum possible repeatability of FA to be 0.64 (Whitlock, 1996). Since observed significant estimates of broad-sense heritability for the FA traits of the finger pattern group vary between 0.20–0.35, the true heritability of underlying developmental stability might be higher, perhaps falling in the 0.31–0.54 range. Further, these heritability estimates overlap considerably with empirically obtained values for the heritability of other anthropometric traits (Chen et al., 1990). These values are even more surprising in the light of numerous studies of morphometric traits that have found FA not to be heritable (Thoday, 1958; Potter and Nance, 1976; Leamy, 1997; Winding, 1998; Corruccini et al., 1988; Corruccini and Potter, 1981). It is possible that, forming during a limited period of time (Penrose and Ohara, 1973; Gooseva, 1986), dermatoglyphic traits, unlike other morphometric traits, do not have much time to be affected by different environmental distortions in their phenotypes. In regard to other morphometric traits, any weak effect of genetic factors is overshadowed by environmental disturbances.

The high genetic diversity of the people living in Moscow may also be responsible for high heritability estimates. The magnitude of fluctuating asymmetry is known to vary among different ethnic groups (Kobyliansky et al., 1979; Ditmar, 1998). Therefore, a genetically mixed population such as Moscow is expected to have high elevated diversity and to produce higher heritability estimates.

The heritability of FA could also be influenced by genetic factors responsible for DA. On the one hand, DA genetic factors could increase the repeatability of FA by determining the direction of the deviation. Asymmetry would thus cease to be directionally random. On the other hand, it is possible that some dermatoglyphic traits are deter-

mined somewhat independently on the left and right hand, or are prone to be more asymmetrical than others. Heritability of FA would then be influenced by the heritability of the dermatoglyphic trait itself rather than the developmental stability.

The fact that genetic factors influence FA more than PME makes FA an ambiguous indicator of developmental stability. The FAs of a-b ridge count and interdigital pattern score would be good candidates for PME indicators, since they are not heritable. Unfortunately, FA indices of these traits do not correlate with the first PC, the only trait in our study that detected a significant PME effect. The combination of traits that did have high loadings on the first PC can be suggested as better indicators of PME.

### CONCLUSIONS

Fluctuating asymmetry indices for the majority of the dermatoglyphic traits in our study had significant broad-sense heritability. The narrow-sense heritability estimates express similar tendencies, but they reach significance in fewer variables.

The observed effect of prenatal environment on FA was weaker than that of genetic factors, and was insignificant for FA of all single dermatoglyphic traits. Weak PME effect may be a result of the small variation in the quality of developmental environment as well as competitive influences of the twins on each other.

The composite scores of FA that are based on a number of dermatoglyphic traits are more useful in detecting genetic and environmental influences, since they solve the problem of the inherent low repeatability of FA. Therefore, FA can be considered a systemic property.

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### LITERATURE CITED

- Arrieta MI, Criado B, Martinez B, Lobato MN, Gil A, Lostao CM. 1993. Fluctuating dermatoglyphic asymmetry: genetic and parental influences. *Ann Hum Biol* 20:557-563.
- Barden HS. 1980. Fluctuating dental asymmetry: a measure of developmental instability in Down syndrome. *Am J Phys Anthropol* 52:169-173.
- Bogle AC, Reed T. 1997. Heritability of dermatoglyphic a-b ridge count asymmetry. *Am J Med Genet* 72:379-380.
- Bogle AC, Reed T, Rose RJ. 1994a. Replication of asymmetry of a-b ridge count and behavioral discordance in monozygotic twins. *Behav Genet* 24:65-72.
- Bogle AC, Reed T, Norton JA Jr. 1994b. Within-pair differences in a-b ridge count asymmetry in monozygotic twins: evidence for placental proximity effect. *Hum Hered* 44:162-168.
- Bracha HS, Torey EF, Gottesman II, Bigelow LB, Cunniff C. 1992. Second-trimester markers of fetal size in schizophrenia: a study of monozygotic twins. *Am J Psychiatry* 149:925-933.
- Chen CJ, Yu MW, Wang CJ, Tong SL, Tien M, Lee TY, Lue HC, Huang FY, Lau CC. 1990. Chronological changes in genetic variance and heritability of anthropometric characteristics among Chinese twin infants. *Acta Genet Med Gemellol (Roma)* 39:479-484.
- Christian JC, Kang KW, Norton JA Jr. 1974. Choice of an estimate of genetic variance from twin data. *Am J Hum Genet* 26:154-161.
- Christian JC, Feinleib M, Norton JA. 1975. Statistical analysis of genetic variance in twins. *Am J Hum Genet* 27:807.
- Corey LA, Nance WE, Kang KW, Christian JC. 1979. Effects of type of placentation on birth weight and its variability in monozygotic and dizygotic twins. *Acta Genet Med Gemellol (Roma)* 28:41-50.
- Corruccini RS, Potter RH. 1981. Developmental correlates of crown component asymmetry and occlusal discrepancy. *Am J Phys Anthropol* 55:21-31.
- Corruccini RS, Sharma K, Potter RHY. 1988. Comparative genetic variance of dental size and asymmetry in U.S. and Punjabi twins. In: Russel DE, Santoro JP, editors. *Teeth revisited: proceedings of the VIIth International Symposium on Dental Morphology*. Paris: Sigogneau-Russel D., Eds. *Mem. Mus NaCN. Hist. Nat., Pairs (ser C)* 53:47-53.
- Cummins H, Midlo C. 1961. *Finger prints, palms and soles. An introduction to dermatoglyphics*. New York: Dover, Inc.
- Ditmar M. 1998. Finger ridge-count asymmetry and diversity in Andean Indians and interpopulation comparisons. *Am J Phys Anthropol* 105:377-393.
- Falconer DS. 1960. *Introduction to quantitative genetics*. New York: Ronald Press Company, Inc. p 169.
- Fraser FC. 1994. Developmental instability and fluctuating asymmetry in man. In: Markow TA, editor. *Developmental instability: its origins and evolutionary implications*. Dordrecht: Kluwer Academic Publishers, Inc. p 319-334.
- Goldberg CJ, Dowling FE, Fogarty EE, Moor DP. 1995. Adolescent idiopathic scoliosis and developmental instability. *Genetica* 96:247-255.
- Goldberg CJ, Fogarty EE, Moor DP, Dowling FE. 1997a. Scoliosis and developmental theory: adolescent idiopathic scoliosis. *Spine* 22:2228-2237.
- Goldberg CJ, Fogarty EE, Moor DP, Dowling FE. 1997b. Fluctuating asymmetry and vertebral malformation.

- A study of dermatoglyphics in congenital spinal deformities. *Spine* 22:775–779.
- Gooseva IS. 1986. Morphogenesis and genetics of ridge skin among humans. Minsk: Belarus Publishers, Inc. [in Russian].
- Gower JC. 1966. Some distance properties of latent root and vector methods used in multivariate analysis. *Biometrika* 53:325–338.
- Green PE. 1976. Mathematical tools for applied multivariate analysis. San Francisco: Academic Press, Inc.
- Haseman JK, Easton RC. 1970. The estimation of genetic variance from twin data. *Behav Genet* 1:11–19.
- Hirth L, Schneider M, Goedde HW. 1984. Papillary ridges of palmar interdigital fields-aspects of bilateral asymmetry [in German]. *Z Morphol Anthropol* 743: 335–343.
- Holt SB. 1954. Genetics of dermal ridges: bilateral asymmetry in finger ridge counts. *Ann Eugen Lond* 18:211–231.
- Jantz RL. 1975. Population variation in asymmetry and diversity from finger to finger for digital ridge-counts. *Am J Phys Anthropol* 42:215–224.
- Kieser JA, Groenvelde HT. 1994. Effects of parental exposure to tobacco smoke on developmental stability in children. *J Craniofac Genet Dev Biol* 14:43–47.
- Kieser JA, Groenvelde HT, Da Silva PCF. 1997. Dental asymmetry, maternal obesity, and smoking. *Am J Phys Anthropol* 102:133–139.
- Kobyliansky E, Micle S, Arensburg B, Nathan H. 1979. Finger dermatoglyphic characteristics in Israeli males. *Z Morphol Anthropol* 70:75–81.
- Kobyliansky E, Bejerano M, Vainder M, Katznelson BM. 1997. Relationship between genetic anomalies of different levels and deviations in dermatoglyphic traits. Part 2: dermatoglyphic peculiarities of females with Turner's syndrome (review). *Anthropol Anz* 55: 315–348.
- Leamy L. 1997. Genetic analysis of fluctuating asymmetry for skeletal characteristics in mice. *J Hered* 88:85–92.
- Livshits G, Kobyliansky E. 1989. Study of genetic variance in the fluctuating asymmetry of anthropometric traits. *Ann Hum Biol* 16:121–130.
- Livshits G, Kobyliansky E. 1991. Fluctuating asymmetry as a possible measure of developmental homeostasis in humans: a review. *Hum Biol* 63:441–466.
- Livshits G, David L, Kobyliansky E, Levi Y, Ben-Amitai D, Merlob P. 1988. Some biological and social factors of risk associated with birth of pre-term infants. *Genet Epidemiol* 5:137–149.
- Livshits G, Yakovenko K, Kletselman L, Karasik D, Kobyliansky D. 1998. Fluctuating asymmetry and morphometric variation of hand bones. *Am J Phys Anthropol* 107:125–136.
- Loesch D, Martin NG. 1982. Directional asymmetry of digital ridge counts. *Acta Anthropogenet* 6:85–98.
- Markow TA, Gottesman II. 1989. Dermatoglyphic fluctuating asymmetry in twins and singletons. *Hereditas* 110:211–215.
- Martin NG, Links JL, Berry HS, Loesh DZ. 1982. A genetical analysis of diversity and asymmetry in finger ridge counts. *Heredity* 48:393–405.
- Mellor CS. 1992. Dermatoglyphic evidence of fluctuating asymmetry in schizophrenia. *Br J Psychiatry* 160: 467–472.
- Møller AP. 1997. Developmental stability, disease and medicine. *Biol Rev Cambridge Philosophic Soc* 72: 497–548.
- Møller AP, Thornhill R. 1997. A meta-analysis of the heritability of developmental stability. *J Ecol Biol* 10:1–16.
- Mooney MP, Siedel MI, Gest TR. 1985. Prenatal stress and increased fluctuating asymmetry in the parietal bones of neonatal rats. *Am J Phys Anthropol* 68:131–134.
- Naugler CT, Ludman MD. 1996a. A case-control study of fluctuating dermatoglyphic asymmetry as risk marker for developmental delay. *Am J Med Genet* 66:11–14.
- Naugler CT, Ludman MD. 1996b. Fluctuating asymmetry and disorders of developmental origin. *Am J Med Genet* 66:15–20.
- Palmer AR. 1994. Fluctuating symmetry analysis: a primer. In: Markow T, editor. *Developmental instability: its origins and evolutionary implications*. Dordrecht: Kluwer Academic Publishers, Inc. p 355–363.
- Palmer AR, Strobeck C. 1997. Fluctuating asymmetry and developmental stability: heritability of observed variation vs. heritability of inferred cause. *Commentary. J Ecol Biol* 10:39–49.
- Penrose LS. 1968. Memorandum on dermatoglyphic nomenclature. *Birth Defects* 4:1–12.
- Penrose LS, Ohara PT. 1973. The development of the epidermal ridges. *Am J Med Genet* 30:185–190.
- Polukhov AM. 1984. The heritability and bimanual asymmetry of finger and palm dermatoglyphics. *Genetica* 20:1894–1901 [in Russian].
- Potter RH, Nance WE. 1976. A twin study of dental dimensions. I. Discordance, asymmetry, and mirror imagery. *Am J Phys Anthropol* 44:391–396.
- Rodewald A, Chopra VP. 1991. Quantitative dermatoglyphic markers in fra-X-syndrome. *Anthropol Anz* 49:121–127.
- Rose RJ, Reed T, Bogle A. 1987. Asymmetry of a-b ridge count and behavioral discordance of monozygotic twins. *Behav Genet* 17:125–140.
- Shapiro B. 1983. Down syndrome—a disruption of homeostasis. *Am J Med Genet* 14:241–269.
- Siegel MI, Smookler HH. 1973. Fluctuating dental asymmetry and auditory stress. *Growth* 37:35–39.
- Siegel MI, Doyle WJ, Kelly C. 1977. Heat stress, fluctuating asymmetry and prenatal selection in the laboratory rat. *Am J Phys Anthropol* 46:121–126.
- Singh S. 1970. Inheritance of asymmetry in finger ridge counts. *Hum Hered* 20:403–408.
- Thoday JM. 1958. Homeostasis in a selection experiment. *Heredity* 12:401–415.
- Thornhill R, Møller AP. 1997. Developmental stability, disease and medicine. *Biol Rev Cambridge Philosophic Soc* 72:497–548.
- Van Valen L. 1962. A study of fluctuating asymmetry. *Evolution* 16:125–142.
- Walpole RE, Myers RH. 1978. Probability and statistics for engineers and scientists, 2nd ed. New York: Macmillan. p 495.
- Whitlock M. 1996. The heritability of fluctuating asymmetry and genetic control of developmental stability. *Proc R Soc Lond [Biol]* 263:849–853.
- Wilkins AS. 1997. Canalization: a molecular genetic perspective. *Bioessays* 19:257–262.
- Winding JJ. 1998. Evolutionary genetics of fluctuating asymmetry in the peacock butterfly. *Heredity* 80:382–392.
- Zakharov VM. 1989. Future prospects for population phenogenetics. *Sov Sci Rev F Physiol Gen Biol* 4:1–7 [in Russian].